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 FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 15:35:41 ON 05 AUG 2004
L1         20415 S "GLUCOCORTICOID RECEPTOR"
L2       1134198 S ANTAGONIST? OR AGONIST?
L3         61892 S SCREENING (S) (METHOD OR PROCESS)
L4          7752 S MMTV OR "MOUSE MAMMARY TUMOR VIRUS"
L5          2251 S "STEROID HORMONE RECEPTOR"
L6          3825 S (HELA OR THP-1 OR THP1) (P) (TNF OR ICAM OR ICAM-1 OR ICAM1 O
L7          4370 S "TYROSINE AMINOTRANSFERASE" OR "TYROSINE TRANSFERASE"
L8        113922 S RAT (S) HEPAT?
L9          3269 S L1 (P) L2
L10            3 S L9 (P) L3
L11            2 DUP REM L10 (1 DUPLICATE REMOVED)
L12          118 S L9 AND L4
L13           96 S L12 NOT PY>=2001
L14           36 DUP REM L13 (60 DUPLICATES REMOVED)
L15            4 S L14 AND L8
L16            4 DUP REM L15 (0 DUPLICATES REMOVED)
L17            0 S L14 AND L7
L18           91 S L9 AND L7
L19           79 S L18 NOT PY>=2001
L20           34 DUP REM L19 (45 DUPLICATES REMOVED)
L21            0 S L6 AND L4 AND L1 AND L2
L22            0 S L6 AND L4 AND L1
L23            0 S L2 AND L6 AND L4
L24         5395 S "TYROSINE AMINOTRANSFERASE" OR "TYROSINE TRANSAMINASE" OR "TY
L25          123 S L24 AND L1 AND L2
L26          596 S TRANSREPRESSION
L27            8 S L25 AND L26
L28            4 DUP REM L27 (4 DUPLICATES REMOVED)
L29          153 S L26 AND L1
L30            0 S L29 AND L3
L31           78 S L29 NOT PY>=2001
L32           35 DUP REM L31 (43 DUPLICATES REMOVED)
L33           34 S L32 NOT PY<=1990
L34            0 S L33 AND L8
L35           54 S L26 AND L1 AND L2
L36           19 S L35 NOT PY>=2001
L37           12 DUP REM L36 (7 DUPLICATES REMOVED)

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ANSWER 1 OF 4 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN

ACCESSION NUMBER: 1998203393 EMBASE
TITLE: Negative regulation of the rat glutathione S-transferase A2 gene by glucocorticoids involves a canonical glucocorticoid consensus sequence.
AUTHOR: Falkner K.C.; Rushmore T.H.; Linder M.W.; Prough R.A.
CORPORATE SOURCE: Dr. R.A. Prough, Dept. of Biochemistry/Molec. Biol., Univ. of Louisville Sch. of Medicine, Louisville, KY 40292, United States. raprou01@ulkyvm.louisville.edu
SOURCE: Molecular Pharmacology, (1998) 53/6 (1016-1026).
Refs: 39
ISSN: 0026-895X CODEN: MOPMA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Glucocorticoids (GCs) repress both basal and polyaromatic hydrocarbon-induced expression of the glutathione S-transferase Ya1 gene (gstA2) in isolated **rat hepatocytes** and **rat** liver in vivo. Transient transfection experiments with HepG2 cells were used to identify GC-responsive elements (GREs). With cotransfected GC receptor, chloramphenicol acetyltransferase (CAT) constructs containing a palindromic GRE (pGRE) and three GRE hexanucleotide half-sites between -1.6 and -1.1 kb of the 5'-flanking region of gstA2 were repressed >50% by GC when induced with polyaromatic hydrocarbon. This pGRE, if either mutated or deleted, significantly reduces GC responsiveness of the gene to 20-30%; no effect of GC was observed with CAT constructs containing -1.15 kb of the 5'-flanking region. The dexamethasone concentration dependence of the repression-was consistent with involvement of the GC receptor and was antagonized by RU38486. Electrophoretic mobility shift assays demonstrated that pGRE formed a specific DNA/protein complex, which was prevented by the addition of excess unlabeled or **mouse mammary tumor virus** GRE but not by unrelated or mutated gstA2 GRE double-stranded oligonucleotides. This complex was supershifted by incubation of nuclear extracts containing GC receptor with anti-GC receptor globulins. Constructs containing multiple copies of pGRE sequence were either nonresponsive or positively responsive (three copies) to GC. Luciferase constructs containing -1.62 to -1.03 kb of the 5'-flanking region also were regulated positively by GC. Chimeric GC-peroxisome proliferator activated receptor activated the constructs that were positively responsive to GC but did not mediate the negative effect in constructs containing 1.6 kb of 5'- flanking region. We conclude that pGRE and half-site GREs of gstA2 participate in regulation of this gene; however, a second unidentified responsive element must exist between -1.03 and -0.164 kb, resulting in repression of gstA2 expression.

L16 ANSWER 2 OF 4 MEDLINE on STN
ACCESSION NUMBER: 87257841 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3037324
TITLE: Glucocorticoid-regulated compartmentalization of cell surface-associated glycoproteins in **rat hepatoma** cells: evidence for an independent response that requires receptor function and de novo RNA synthesis.
AUTHOR: Haffar O K; Vallerga A K; Marenda S A; Witchel H J; Firestone G L
CONTRACT NUMBER: CA 09041 (NCI)
CA 35547 (NCI)
SOURCE: Molecular and cellular biology, (1987 Apr) 7 (4) 1508-17.
Journal code: 8109087. ISSN: 0270-7306.

PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198707
ENTRY DATE: Entered STN: 19900305
Last Updated on STN: 19970203
Entered Medline: 19870724

AB The role of glucocorticoid hormones in the compartmentalization of cell surface-associated **mouse mammary tumor virus** (**MMTV**) glycoproteins was examined in M1.54, a cloned line of **MMTV**-infected **rat hepatoma** tissue culture cells. The expression of cellular [2-3H]mannose-labeled and cell surface 125I-labeled **MMTV** glycoproteins was examined throughout a time course of exposure to dexamethasone, a synthetic glucocorticoid. Posttranslational localization of cell surface **MMTV** glycoproteins required 6 h of exposure to hormone and occurred approximately 4 h after their initial production in an intracellular fraction. This regulated localization to the cell surface correlated with **glucocorticoid receptor** occupancy and was inhibited by exposure to RU 38486, a powerful **antagonist** of glucocorticoid-mediated responses. Cell surface immunoprecipitation demonstrated that actinomycin D, an inhibitor of de novo RNA synthesis, prevented regulated expression of cell surface viral glycoproteins, suggesting that newly synthesized cellular components mediate this process. The localization of cell surface **MMTV** glycoproteins appeared normal in a transcriptional variant (CR1) that produces basal levels of **MMTV** RNA and glycoprotein precursors in the presence of dexamethasone. Thus, regulated compartmentalization of viral glycoproteins is not an obligate consequence of a critical precursor concentration. Taken together, our results suggest that posttranslational trafficking of cell surface-destined **MMTV** glycoproteins resulted from an independent glucocorticoid hormone response that required receptor function and de novo RNA synthesis.

L16 ANSWER 3 OF 4 MEDLINE on STN
ACCESSION NUMBER: 91042568 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2856402
TITLE: Dual regulation of protein maturation in viral infected **rat** HTC **hepatoma** cells by glucocorticoids and progesterone.
AUTHOR: Winguth S D; Firestone G L
CORPORATE SOURCE: Department of Physiology-Anatomy, University of California, Berkeley 94720.
CONTRACT NUMBER: CA-09041 (NCI)
SOURCE: Molecular endocrinology (Baltimore, Md.), (1987 Nov) 1 (11) 823-33.
Journal code: 8801431. ISSN: 0888-8809.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199012
ENTRY DATE: Entered STN: 19910208
Last Updated on STN: 19970203
Entered Medline: 19901205

AB Dexamethasone, a synthetic glucocorticoid, is required for full posttranslational maturation of **mouse mammary tumor virus** (**MMTV**) phosphoproteins and glycoproteins in M1.54 cells, a viral infected **rat hepatoma** (HTC) cell line. Pulse-chase radiolabeling with [35S]methionine revealed that steroids with known glucocorticoid activity (such as dexamethasone and hydrocortisone) regulated the maturation of

both **MMTV** polyproteins in a manner proportional to their occupancy for glucocorticoid receptors and their biological potency. In contrast, progesterone selectively induced the proteolytic processing of **MMTV** phosphoproteins but simultaneously antagonized the dexamethasone-regulated maturation of **MMTV** glycoproteins and all other tested glucocorticoid responses. Exposure to suboptimal concentrations of both progesterone and dexamethasone fully stimulated the processing of **MMTV** phosphoproteins, suggesting that steroid receptors occupied with combinations of either steroid functionally interact at the putative maturation gene. Moreover, treatment with either actinomycin D, a potent inhibitor of de novo RNA synthesis, or RU38486, a synthetic **antagonist** of glucocorticoid and progesterone action, prevented both the dexamethasone and progesterone-regulated induction of **MMTV** phosphoprotein maturation. Sedimentation velocity and saturation binding analysis revealed that the sizes and concentrations of hepatoma cell progesterone and dexamethasone binding activities are similar while specific binding of the active progestin R5020 was not detected in either M1.54 cells or the **glucocorticoid receptor** deficient HTC cell line MSN6.10.2. Taken together, our results demonstrate that two distinct classes of steroid hormones can uniquely alter the posttranslational maturation of a specific subset of phosphoprotein substrates by a common **glucocorticoid receptor**-dependent process.

L16 ANSWER 4 OF 4 MEDLINE on STN
ACCESSION NUMBER: 84057145 MEDLINE
DOCUMENT NUMBER: PubMed ID: 6139269
TITLE: Induction of the acute-phase reactant, alpha 1-acid glycoprotein, by glucocorticoids in **rat hepatoma** cells.
AUTHOR: Vannice J L; Ringold G M; McLean J W; Taylor J M
CONTRACT NUMBER: CA33563 (NCI)
GM07149 (NIGMS)
GM25821 (NIGMS)
+
SOURCE: DNA (Mary Ann Liebert, Inc.), (1983) 2 (3) 205-12.
Journal code: 8302432. ISSN: 0198-0238.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198401
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19970203
Entered Medline: 19840107

AB alpha 1-acid glycoprotein (alpha 1-AGP), or orosomucoid, is shown to be inducible by glucocorticoids in HTC **rat hepatoma** cells. Immunoprecipitation of [35S]methionine pulse-labeled proteins from these cells reveals secreted proteins of Mr = 35,000-48,000 (alpha 1-AGP) and Mr greater than 180,000, both of which are greatly enhanced by glucocorticoid treatment. The amount of alpha 1-AGP-specific mRNA in HTC cells is greatly increased (at least 100-fold) in response to glucocorticoids. The new steady-state level of RNA is approached with a t 1/2 of about 8 hr and the RNA consists of a single species of approximately 850 bases. The response is specific for glucocorticoids since: (i) the EC50 for dexamethasone is 30 nM; (ii) the glucocorticoid **antagonist**, progesterone, inhibits the induction by dexamethasone; and (iii) a **glucocorticoid receptor**-deficient cell line is incapable of alpha 1-AGP mRNA induction. This is a secondary hormonal response since inhibition of protein synthesis blocks the induction of alpha 1-AGP mRNA by dexamethasone whereas the induction of **mouse mammary tumor virus** (**MMTV**) RNA is unaffected.

L37 ANSWER 1 OF 12 MEDLINE on STN
ACCESSION NUMBER: 2000482932 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10974006
TITLE: Glucocorticoids antagonize AP-1 by inhibiting the Activation/phosphorylation of JNK without affecting its subcellular distribution.
AUTHOR: Gonzalez M V; Jimenez B; Berciano M T; Gonzalez-Sancho J M; Caelles C; Lafarga M; Munoz A
CORPORATE SOURCE: Instituto de Investigaciones Biomedicas Alberto Sols, Consejo Superior de Investigaciones Cientificas-Universidad Autonoma de Madrid, E-28029 Madrid, Spain.
SOURCE: Journal of cell biology, (2000 Sep 4) 150 (5) 1199-208. Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200010
ENTRY DATE: Entered STN: 20001019
Last Updated on STN: 20001019
Entered Medline: 20001010

AB The immunosuppressive and antiinflammatory actions of glucocorticoid hormones are mediated by their **transrepression** of activating protein-1 (AP-1) and nuclear factor-kappa B (NFkappaB) transcription factors. Inhibition of the c-Jun NH(2)-terminal kinase (JNK) signaling pathway, the main mediator of AP-1 activation, has been described in extracts of hormone-treated cells. Here, we show by confocal laser microscopy, enzymatic assays, and immunoblotting that the synthetic glucocorticoid dexamethasone inhibited tumor necrosis factor alpha (TNF-alpha)-induced phosphorylation and activation of JNK in the cytoplasm and nucleus of intact HeLa cells. As a result, c-Jun NH(2)-terminal domain phosphorylation and induction were impaired. Dexamethasone did not block the TNF-alpha-induced JNK nuclear translocation, but rather induced, per se, nuclear accumulation of the enzyme. Consistently with previous findings, a **glucocorticoid receptor** mutant (GRdim), which is deficient in dimerization, DNA binding, and transactivation, but retains AP-1 transrepressing activity, was as efficient as wild-type GR in mediating the same effects of dexamethasone on JNK in transfected Cos-7 cells. Our results show that glucocorticoids antagonize the TNF-alpha-induced activation of AP-1 by causing the accumulation of inactive JNK without affecting its subcellular distribution.

L37 ANSWER 2 OF 12 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2000425239 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10948677
TITLE: Molecular mechanisms of corticosteroid actions.
AUTHOR: Adcock I M; Ito K
CORPORATE SOURCE: Dept of Thoracic Medicine, National Heart and Lung Institute, Imperial College School of Medicine, London, UK.
SOURCE: Monaldi archives for chest disease = Archivio Monaldi per le malattie del torace / Fondazione clinica del lavoro, IRCCS [and] Istituto di clinica tisiologica e malattie apparato respiratorio, Universita di Napoli, Secondo ateneo, (2000 Jun) 55 (3) 256-66. Ref: 103 Journal code: 9307314. ISSN: 1122-0643.
PUB. COUNTRY: Italy
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200009

ENTRY DATE: Entered STN: 20000922
Last Updated on STN: 20000922
Entered Medline: 20000908

AB Corticosteroids are the most effective therapy for the treatment of inflammatory diseases such as asthma. Functionally, they act partly by inducing anti-inflammatory genes such as secretary leukocyte proteinase inhibitor, Lipocortin-1 and interleukin-1 receptor **antagonist**, but mainly by repression of inflammatory genes, such as cytokines, adhesion molecules, inflammatory enzymes and receptors. They act by binding to a cytosolic **glucocorticoid receptor** (GR), which upon binding is activated and rapidly translocates to the nucleus. Within the nucleus, the GR either induces gene transcription by binding to specific deoxyribonucleic acid elements in the promoter/enhancer regions of responsive genes or reduces gene transcription by **transrepression**. The GR reduces gene transcription by interaction with pro-inflammatory transcription factors such as activation protein-1 and nuclear factor-kappa B. These effects of the GR on gene expression involve changes in the chromatin structure localized to the promoter sites of responsive genes. Many of the detrimental side-effects of corticosteroids are believed to be due to gene induction, leading to the search for novel corticosteroids which can repress inflammatory genes without inducing gene transcription.

L37 ANSWER 3 OF 12 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2000017941 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10551779
TITLE: Structure/function of the human **glucocorticoid receptor**: tyrosine 735 is important for transactivation.
AUTHOR: Ray D W; Suen C S; Brass A; Soden J; White A
CORPORATE SOURCE: Department of Medicine, University of Manchester, United Kingdom.. DRAY@fs1.scg.man.ac.uk
SOURCE: Molecular endocrinology (Baltimore, Md.), (1999 Nov) 13 (11) 1855-63.
Journal code: 8801431. ISSN: 0888-8809.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199911
ENTRY DATE: Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991130

AB Ligand-induced activation of the **glucocorticoid receptor** (GR) is not well understood. The GR ligand-binding domain was modeled, based on homology with the progesterone receptor. Tyrosine 735 interacts with the D ring of dexamethasone, and substitution of D ring functional groups results in partial **agonist** steroids with reduced ability to direct transactivation. Loss of the Tyr735 hydroxyl group by substitution to phenylalanine (Tyr735Phe) did not reduce ligand binding affinity [dissociation constant (Kd) 4.3 nM compared with Kd 4.6 nM for wild-type] and did not alter **transrepression** of an nuclear factor-kappaB (NF-kappaB reporter. But, there was a significant 30% reduction in maximal transactivation of a mouse mammary tumor virus (MMTV) reporter, although with an unchanged EC50 (8.6 nM compared with 6 nM). Substitution to a nonaromatic hydrophobic amino acid, valine (Tyr735Val), retained high-affinity ligand binding for dexamethasone (Kd 6 nM compared with 4.6 nM) and did not alter **transrepression** of NF-kappaB. However, there was a 36% reduction in MMTV activity with a right shift in EC50 (14.8 nM). The change to serine, a small polar amino acid (Tyr735Ser), caused significantly lower affinity for dexamethasone (10.4 nM). Maximal **transrepression** of NF-kappaB was unaltered, but the IC50 for this effect was increased. Tyr735Ser had a major shift in

EC50 (118 nM) for transactivation of an MMTV reporter. Maximal transactivation of MMTV induced by the natural ligand cortisol was reduced to 60% by Tyr735Phe and Tyr735Val and was completely absent by Tyr735Ser. These data suggest that tyrosine 735 is important for ligand interpretation and transactivation.

L37 ANSWER 4 OF 12 MEDLINE on STN
ACCESSION NUMBER: 1999426965 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10496964
TITLE: Dissociated glucocorticoids with anti-inflammatory potential repress interleukin-6 gene expression by a nuclear factor-kappaB-dependent mechanism.
AUTHOR: Vanden Berghe W; Francesconi E; De Bosscher K; Resche-Rigon M; Haegeman G
CORPORATE SOURCE: Department of Molecular Biology University of Gent and Flanders Interuniversity Institute for Biotechnology, Gent, Belgium.
SOURCE: Molecular pharmacology, (1999 Oct) 56 (4) 797-806. Journal code: 0035623. ISSN: 0026-895X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199910
ENTRY DATE: Entered STN: 19991026
Last Updated on STN: 20020919
Entered Medline: 19991008

AB Synthetic glucocorticoids (GCs) remain among the most effective agents for the management of chronic inflammatory diseases. However, major side effects severely limit their therapeutic use. Physiologic and therapeutic activities of GCs are mediated by a nuclear receptor belonging to a superfamily of ligand-inducible transcription factors that, in addition to directly regulating their cognate gene programs, can also mutually interfere with other signaling pathways. We recently identified selective ligands of the **glucocorticoid receptor** that dissociate transactivation from activator protein 1 **transrepression**, and most importantly retain in vivo anti-inflammatory activity. To further document the mechanisms of action sustaining the observed in vivo activity, we report here on the interference of dissociated GCs with nuclear factor kappaB (NF-kappaB)-driven gene activation. We show that dissociated GCs repress tumor necrosis factor-induced interleukin-6 gene expression by an NF-kappaB-dependent mechanism, without changing the expression level of inhibitor kappaB. The DNA-binding activity of induced NF-kappaB also remained unchanged after stimulation of cells with the various compounds. Evidence for a direct nuclear mechanism of action was obtained by analysis of cell lines constitutively expressing a fusion protein between the DNA-binding domain of the yeast Gal4 protein and the transactivating p65 subunit of NF-kappaB, which was able to efficiently repress a Gal4-dependent luciferase reporter gene upon addition of the dissociated compounds. We therefore conclude that, in addition to dissociating transactivation from activator protein 1 **transrepression**, dissociated GCs mediate inhibition of NF-kappaB signaling by a mechanism that is independent of inhibitor kappaB induction.

L37 ANSWER 5 OF 12 MEDLINE on STN
ACCESSION NUMBER: 2000084419 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10619401
TITLE: Interaction of **glucocorticoid receptor** isoforms with transcription factors AP-1 and NF-kappaB: lack of effect of **glucocorticoid receptor** beta.
AUTHOR: Brogan I J; Murray I A; Cerillo G; Needham M; White A;

Davis J R
CORPORATE SOURCE: School of Biological Sciences, University of Manchester, UK.
SOURCE: Molecular and cellular endocrinology, (1999 Nov 25) 157 (1-2) 95-104.
Journal code: 7500844. ISSN: 0303-7207.
PUB. COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 20000218
Last Updated on STN: 20000218
Entered Medline: 20000210

AB Glucocorticoids act through the **glucocorticoid receptor** (GR) to enhance or repress transcription of glucocorticoid responsive genes depending on the promoter context and cellular background. The human GR primary transcript is alternatively spliced resulting in hGR alpha and hGR beta isoforms. Transactivation and **transrepression** are mediated by hGR alpha and while it has been demonstrated that hGR beta, can act as a dominant negative inhibitor of hGR alpha mediated transactivation, its effects on **transrepression** are not known. To investigate hGR beta actions, we used GR-deficient COS-7 and HEK-293 cells. When hGR alpha (0.5 microg 10(6) cells(-1)) was transfected into COS-7 cells dexamethasone (150 nM) inhibited TNF alpha (80 U ml(-1)) effects on a NF-kappaB responsive reporter gene by 40%. There was no evidence of a dominant negative effect when hGR beta (1-10 microg) was co-transfected with hGR alpha up to ratios of 10:1. Similarly hGR beta had no effect on hGR alpha inhibition of a phorbol ester stimulated Ap-1-responsive reporter gene in COS-7 or HEK-293 cells. In comparison, an apparent dominant negative effect of hGR beta on hGR alpha-mediated transactivation was found to be attributable to non-specific transcriptional squelching in COS-7 cells. In summary, the potential for hGR beta, to act as a dominant negative inhibitor of hGR alpha-mediated transactivation remains controversial, but our data suggest that hGR beta, was unable to act as a dominant negative inhibitor of either hGR alpha-mediated **transrepression** or transactivation in these promoter and cell contexts.

L37 ANSWER 6 OF 12 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1999:392460 BIOSIS
DOCUMENT NUMBER: PREV199900392460
TITLE: Identification of a glucocorticoid that dissociates transactivation from **transrepression** in normal human lymphocytes.
AUTHOR(S): Bamberger, C. M. [Reprint author]; Else, T. [Reprint author]; Bamberger, A. M. [Reprint author]; Beil, F. U.; Schulte, H. M. [Reprint author]
CORPORATE SOURCE: IHF Institute for Hormone and Fertility Research, University of Hamburg, Hamburg, Germany
SOURCE: European Journal of Clinical Investigation, (April, 1999) Vol. 29, No. SUPPL. 1, pp. 19. print.
Meeting Info.: 33rd Meeting of the European Society for Clinical Investigation. Milan, Italy. April 8-10, 1999. European Society for Clinical Investigation.
CODEN: EJCIB8. ISSN: 0014-2972.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 28 Sep 1999
Last Updated on STN: 28 Sep 1999

L37 ANSWER 7 OF 12 EMBASE

on STN

ACCESSION NUMBER: 1998194671 EMBASE

TITLE: Anti-inflammatory actions of glucocorticoids: Molecular mechanisms.

AUTHOR: Barnes P.J.

CORPORATE SOURCE: Prof. P.J. Barnes, Department of Thoracic Medicine, National Heart and Lung Institute, Imperial College, Dovehouse St, London SW3 6LY, United Kingdom

SOURCE: Clinical Science, (1998) 94/6 (557-572).
Refs: 178
ISSN: 0143-5221 CODEN: CSCIAE

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 005 General Pathology and Pathological Anatomy
030 Pharmacology
031 Arthritis and Rheumatism
037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

AB 1. Glucocorticoids are widely used for the suppression of inflammation in chronic inflammatory diseases such as asthma, rheumatoid arthritis, inflammatory bowel disease and autoimmune diseases, all of which are associated with increased expression of inflammatory genes. The molecular mechanisms involved in this anti-inflammatory action of glucocorticoids is discussed, particularly in asthma, which accounts for the highest clinical use of these agents. 2. Glucocorticoids bind to glucocorticoid receptors in the cytoplasm which then dimerize and translocate to the nucleus, where they bind to glucocorticoid response elements (GRE) on glucocorticoid-responsive genes, resulting in increased transcription. Glucocorticoids may increase the transcription of genes coding for anti-inflammatory proteins, including lipocortin-1, interleukin-10, interleukin-1 receptor **antagonist** and neutral endopeptidase, but this is unlikely to account for all of the widespread anti-inflammatory actions of glucocorticoids. 3. The most striking effect of glucocorticoids is to inhibit the expression of multiple inflammatory genes (cytokines, enzymes, receptors and adhesion molecules). This cannot be due to a direct interaction between glucocorticoid receptors and GRE, as these binding sites are absent from the promoter regions of most inflammatory genes. It is more likely to be due to a direct inhibitory interaction between activated glucocorticoid receptors and activated transcription factors, such as nuclear factor-κB and activator protein-1, which regulate the inflammatory gene expression. 4. It is increasingly recognized that glucocorticoids change the chromatin structure. Glucocorticoid receptors also interact with CREB-binding protein (CBP), which acts as a co-activator of transcription, binding several other transcription factors that compete for binding sites on this molecule. Increased transcription is associated with uncoiling of DNA wound around histone and this is secondary to acetylation of the histone residues by the enzymic action of CBP. Glucocorticoids may lead to deacetylation of histone, resulting in tighter coiling of DNA and reduced access of transcription factors to their binding sites, thereby suppressing gene expression. 5. Rarely patients with chronic inflammatory diseases fail to respond to glucocorticoids, although endocrine function of steroids is preserved. This may be due to excessive formation of activator protein-1 at the inflammatory site, which consumes activated glucocorticoid receptors so that they are not available for suppressing inflammatory genes. 6. This new understanding of glucocorticoid mechanisms may lead to the development of novel steroids with less risk of side effects (which are due to the endocrine and metabolic actions of steroids). 'Dissociated' steroids which are more active in **transrepression** (interaction with transcription factors) than transactivation (GRE binding) have now been developed. Some of the transcription factors that are inhibited by glucocorticoid, such as nuclear factor-κB, are also targets for

novel anti-inflammatory therapies.

L37 ANSWER 8 OF 12 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 1999107005 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9891987
TITLE: Various glucocorticoids differ in their ability to induce gene expression, apoptosis and to repress NF-kappaB-dependent transcription.
AUTHOR: Hofmann T G; Hehner S P; Bacher S; Droge W; Schmitz M L
CORPORATE SOURCE: German Cancer Research Center (DKFZ), Department of Immunochemistry, Heidelberg.
SOURCE: FEBS letters, (1998 Dec 28) 441 (3) 441-6.
Journal code: 0155157. ISSN: 0014-5793.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199902
ENTRY DATE: Entered STN: 19990223
Last Updated on STN: 19990223
Entered Medline: 19990205

AB Glucocorticoids (GCs) influence a great variety of cellular functions by at least three important modes of action: the activation (or repression) of genes controlled by binding sites for the **glucocorticoid receptor** (GR), the induction of apoptosis in lymphocytes and the recently discovered cross-talk to other transcription factors such as NF-kappaB. In this study we systematically compared various natural and synthetic steroid hormones frequently used as therapeutic agents on their ability to mediate these three modes of action. Betamethasone, triamcinolone, dexamethasone and clobetasol turned out to be the best inducers of gene expression and apoptosis. All GCs including the **antagonistic** compound RU486 efficiently reduced NF-kappaB-mediated transactivation to comparable extents, suggesting that ligand-induced nuclear localization of the GR is sufficient for **transrepression** . Glucocorticoid treatment of cells did not result in elevated IkappaB-alpha expression, but impaired the tumor necrosis factor (TNF)-alpha-induced degradation of IkappaB-alpha without affecting DNA binding of NF-kappaB. The structural requirements for the various functions of glucocorticoids are discussed.

L37 ANSWER 9 OF 12 MEDLINE on STN
ACCESSION NUMBER: 1998227960 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9569005
TITLE: Modulation of hormone-dependent **glucocorticoid receptor** function using a tetracycline-regulated expression system.
AUTHOR: Wei P; Ahn Y I; Housley P R; Alam J; Vedeckis W V
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, Louisiana State University Medical Center, New Orleans 70112, USA.
CONTRACT NUMBER: DK43 135 (NIDDK)
DK47211 (NIDDK)
DK47951 (NIDDK)
SOURCE: Journal of steroid biochemistry and molecular biology, (1998 Jan) 64 (1-2) 1-12.
Journal code: 9015483. ISSN: 0960-0760.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199805
ENTRY DATE: Entered STN: 19980520
Last Updated on STN: 19980520
Entered Medline: 19980513

AB The **glucocorticoid receptor** (GR) is a ligand-dependent transcription factor capable of stimulating and inhibiting the expression of target genes. To better understand the biological action of glucocorticoids and the function of GR, we have utilized the tetracycline (Tc)-regulated mammalian expression system to develop a novel cell line, E8.2/GR3, derived from GR null mouse L929 fibroblasts, that exhibits conditional expression of rat GR. The intracellular concentration of rGR in E8.2/GR3 cells--from undetectable levels to levels more than 10-fold greater than that observed in wild-type L929 cells--could be manipulated by varying the Tc concentration in the culture media. Similarly, dexamethasone (DEX)-dependent transactivation of the mouse mammary tumor virus long terminal repeat and **transrepression** of the cadmium-induced activity of the mouse heme oxygenase-1 gene enhancer, SX2, were strictly dependent on the presence of rGR, and the levels of these activities could be modulated by Tc. Similar levels of Tc, and thus rGR, were required for half-maximal transactivation and **transrepression** whereas a 6-fold lower concentration of DEX was required for half-maximal **transrepression** than for transactivation. RU486 inhibited both DEX-dependent transactivation and **transrepression**. DEX decreased the steady-state level of rGR mRNA and protein in a Tc dependent manner. DEX also induced morphological changes in E8.2/GR3 cells that were dependent on rGR as no alterations were observed in the presence of Tc. These cells provide a powerful system for examining the various activities of GR, particularly as a function of different intracellular receptor concentrations.

L37 ANSWER 10 OF 12 MEDLINE on STN
ACCESSION NUMBER: 97407940 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9261164
TITLE: A new function for the C-terminal zinc finger of the **glucocorticoid receptor**. Repression of RelA transactivation.
AUTHOR: Liden J; Delaunay F; Rafter I; Gustafsson J; Okret S
CORPORATE SOURCE: Department of Medical Nutrition, Karolinska Institute, Huddinge University Hospital, Novum F60, S-141 86 Huddinge, Sweden.
SOURCE: Journal of biological chemistry, (1997 Aug 22) 272 (34) 21467-72.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199709
ENTRY DATE: Entered STN: 19970926
Last Updated on STN: 20000303
Entered Medline: 19970915

AB Glucocorticoids inhibit NF-kappaB signaling by interfering with the NF-kappaB transcription factor RelA. Previous studies have identified the DNA-binding domain (DBD) in the **glucocorticoid receptor** (GR) as the major region responsible for this repressive activity. Using GR mutants with chimeric DBDs the repressive function was found to be located in the C-terminal zinc finger. As predicted from these results the mineralocorticoid receptor that contains a C-terminal zinc finger identical to that of the GR was also able to repress RelA-dependent transcription. Mutation of a conserved arginine or a lysine in the second zinc finger of the GR DBD (Arg-488 or Lys-490 in the rat GR) abolished the ability of GR to inhibit RelA activity. In contrast, C-terminal zinc finger GR mutants with mutations in the dimerization box or mutations necessary for full transcriptional GR activity were still able to repress RelA-dependent transcription. In addition, we found that the steroid analog ZK98299 known to induce GR **transrepression** of AP-1 had no inhibitory effect on RelA activity. In summary, these results demonstrate

that the inhibition of NF-kappaB by glucocorticoids involves two critical amino acids in the C-terminal zinc finger of the GR. Furthermore, the results from the use of mineralocorticoid receptor and anti-glucocorticoids suggest that the mechanisms for GR-mediated repression of NF-kappaB and AP-1 are different.

L37 ANSWER 11 OF 12 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 95124304 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7823916
TITLE: Hormone-independent repression of AP-1-inducible collagenase promoter activity by glucocorticoid receptors.
AUTHOR: Liu W; Hillmann A G; Harmon J M
CORPORATE SOURCE: Department of Pharmacology, Uniformed Services University of the Health Sciences, Bethesda, Maryland 20814-4799.
CONTRACT NUMBER: CA32226 (NCI)
SOURCE: Molecular and cellular biology, (1995 Feb) 15 (2) 1005-13. Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 19950223
Last Updated on STN: 19980206
Entered Medline: 19950216

AB The role of the ligand in **glucocorticoid receptor** -mediated transactivation and **transrepression** of gene expression was investigated. Half-maximal transactivation of a mouse mammary tumor virus-chloramphenicol acetyltransferase reporter gene in transfected cells expressing the human **glucocorticoid receptor** mutant GRL753F, from which the rate of ligand dissociation is four to five times higher than the rate of dissociation from normal receptors, required a 200- to 300-fold-higher concentration of dexamethasone than was required in cells expressing the normal receptor. Immunocytochemical analysis demonstrated that this difference was not the result of a failure of the mutant receptor to accumulate in the nucleus after steroid treatment. In contrast, in cells cotransfected with a reporter gene containing the AP-1-inducible collagenase gene promoter, the concentration of dexamethasone required for 50% **transrepression** was the same for mutant and normal receptors. Efficient receptor-mediated **transrepression** was also observed with the double mutant GRL753F/C421Y, in which the first cysteine residue of the proximal zinc finger has been replaced by tyrosine, indicating that neither retention of the ligand nor direct binding of the receptor to DNA is required. RU38486 behaved as a full **agonist** with respect to **transrepression**. In addition, receptor-dependent **transrepression**, but not transactivation, was observed in transfected cells after heat shock in the absence of the ligand. Taken together, these results suggest that unlike transactivation, **transrepression** of AP-1 activity by the nuclear **glucocorticoid receptor** is ligand independent.

L37 ANSWER 12 OF 12 MEDLINE on STN
ACCESSION NUMBER: 95124352 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7823959
TITLE: Characterization of mechanisms involved in **transrepression** of NF-kappa B by activated glucocorticoid receptors.
AUTHOR: Scheinman R I; Gualberto A; Jewell C M; Cidlowski J A; Baldwin A S Jr
CORPORATE SOURCE: Lineberger Comprehensive Cancer Center, University of North Carolina at Chapel Hill 27599.
SOURCE: Molecular and cellular biology, (1995 Feb) 15 (2) 943-53.

Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 19950223
Last Updated on STN: 19980206
Entered Medline: 19950216

AB Glucocorticoids are potent immunosuppressants which work in part by inhibiting cytokine gene transcription. We show here that NF-kappa B, an important regulator of numerous cytokine genes, is functionally inhibited by the synthetic glucocorticoid dexamethasone (DEX). In transfection experiments, DEX treatment in the presence of cotransfected **glucocorticoid receptor** (GR) inhibits NF-kappa B p65-mediated gene expression and p65 inhibits GR activation of a glucocorticoid response element. Evidence is presented for a direct interaction between GR and the NF-kappa B subunits p65 and p50. In addition, we demonstrate that the ability of p65, p50, and c-rel subunits to bind DNA is inhibited by DEX and GR. In HeLa cells, DEX activation of endogenous GR is sufficient to block tumor necrosis factor alpha or interleukin 1 activation of NF-kappa B at the levels of both DNA binding and transcriptional activation. DEX treatment of HeLa cells also results in a significant loss of nuclear p65 and a slight increase in cytoplasmic p65. These data reveal a second mechanism by which NF-kappa B activity may be regulated by DEX. We also report that RU486 treatment of wild-type GR and DEX treatment of a transactivation mutant of GR each can significantly inhibit p65 activity. In addition, we found that the zinc finger domain of GR is necessary for the inhibition of p65. This domain is also required for GR repression of AP-1. Surprisingly, while both AP-1 and NF-kappa B can be inhibited by activated GR, synergistic NF-kappa B/AP-1 activity is largely unaffected. These data suggest that NF-kappa B, AP-1, and GR interact in a complex regulatory network to modulate gene expression and that cross-coupling of NF-kappa B and GR plays an important role in glucocorticoid-mediated repression of cytokine transcription.

L Number	Hits	Search Text	DB	Time stamp
-	2205	"glucocorticoid receptor"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 13:42
-	1	"dissociated glucocorticoid receptor"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 13:43
-	78453	antagonist or agonist	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 13:43
-	1104	"glucocorticoid receptor" and (antagonist or agonist)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 13:43
-	0	"method for screening" or "method of screening"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 13:44
-	44861	screening WITH method	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 13:44
-	18243	screening WITH process	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 13:44
-	3793	(antagonist or agonist) SAME (screening WITH method)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 13:44
-	76	((antagonist or agonist) SAME (screening WITH method)) and "glucocorticoid receptor"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 13:50
-	253	"glucocorticoid receptor" WITH (antagonist or agonist)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 13:50
-	4	("glucocorticoid receptor" WITH (antagonist or agonist)) SAME (screening WITH method)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 14:27
-	1	("glucocorticoid receptor" WITH (antagonist or agonist)) SAME (screening WITH process)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 14:40
-	256	"tyrosine aminotransferase"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 14:43
-	2936	(MMTV or "mouse mammary tumor virus")SAME promoter	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 14:45
-	1	(("glucocorticoid receptor" WITH (antagonist or agonist)) SAME (screening WITH method)) and "tyrosine aminotransferase"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 14:45
-	3	(("glucocorticoid receptor" WITH (antagonist or agonist)) SAME (screening WITH method)) and ((MMTV or "mouse mammary tumor virus")SAME promoter)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 14:46
-	3877	thomson.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 14:46

-	3877	thomson.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 14:46
-	21	thomson.in. and "glucocorticoid receptor"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 14:47
-	10797	boehringer.as.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 14:47
-	33	boehringer.as. and "glucocorticoid receptor"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 14:48
-	91	jennewein.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 14:48
-	2	jennewein.in. and "glucocorticoid receptor"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 14:58
-	1000	Mcdonnell.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 14:58
-	5	Mcdonnell.in. and "glucocorticoid receptor"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 15:01
-	1476	"steroid hormone receptor"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 15:01
-	135	"steroid hormone receptor" sAME (antagonist or agonist)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 15:02
-	19	("steroid hormone receptor" sAME (antagonist or agonist)) and ((screening WITH method) or (screening WITH process))	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 15:06
-	4	5902732.pn. or 5876946.pn.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 15:14
-	27215	TNF-alpha or TNF or ICAM-1 or (LPS SAME IL-8)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 15:14
-	515	(TNF-alpha or TNF or ICAM-1 or (LPS SAME IL-8)) and "glucocorticoid receptor"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 15:14
-	21655	heLa or THP1	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 15:15
-	114	(((TNF-alpha or TNF or ICAM-1 or (LPS SAME IL-8)) and "glucocorticoid receptor") and (antagonist or agonist)) and (heLa or THP1)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 15:16
-	70	((((TNF-alpha or TNF or ICAM-1 or (LPS SAME IL-8)) and "glucocorticoid receptor") and (antagonist or agonist)) and (heLa or THP1)) and ((screening WITH method) or (screening WITH process))	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 15:27

-	4954	rat WITH hepat$	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 15:27
-	75	(rat WITH hepat$) and "tyrosine aminotransferase"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 15:27
-	18	((rat WITH hepat$) and "tyrosine aminotransferase") and "glucocorticoid receptor"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 15:28
-	272	((TNF-alpha or TNF or ICAM-1 or (LPS SAME IL-8)) and "glucocorticoid receptor") and (antagonist or agonist)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 16:13
-	276	"tyrosine aminotransferase" or "tyrosine transferase" or "tyrosine transaminase"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 16:14
-	61	("tyrosine aminotransferase" or "tyrosine transferase" or "tyrosine transaminase") and "glucocorticoid receptor"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 16:14
-	141	transrepression	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 16:14
-	9	(("tyrosine aminotransferase" or "tyrosine transferase" or "tyrosine transaminase") and "glucocorticoid receptor") and transrepression	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 16:19
-	310	Ligand$.as.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 16:19
-	78	Ligand$.as. and "glucocorticoid receptor"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 16:19
-	65	(Ligand$.as. and "glucocorticoid receptor") and (antagonist or agonist)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 16:20
-	1	((Ligand$.as. and "glucocorticoid receptor") and (antagonist or agonist)) and Mcdonnell.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 16:21
-	0	((Ligand$.as. and "glucocorticoid receptor") and (antagonist or agonist)) and transrepression	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/05 16:22

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3	US 20040110154 A1	Method for creating specific, high affinity nuclear receptor pharmaceuticals
4	US 20040097707 A1	Receptors and membrane-associated proteins
5	US 20040086896 A1	Polynucleotides and polypeptides associated with the NF-kB pathway
6	US 20040077010 A1	32142, 21481, 25964, 21686, novel dehydrogenase molecules and uses therefor
7	US 20040068762 A1	Transgenic non-human mammals expressing a reporter nucleic acid under the regulation of androgen response elements
8	US 20040059089 A1	Coativation of nuclear receptors
9	US 20040053834 A1	Enzymes involved in glycoprotein and glycolipid metabolism
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13	US 20040009558 A1	Ligand binding pocket peptide fragment of the estrogen-related receptor 3 (ERR3) and uses thereof
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15	US 20030224390 A1	Method of identifying conformation-sensitive binding peptides and uses thereof
16	US 20030219832 A1	Synthesis and use of retinoid compounds having negative hormone and/or antagonist activities
17	US 20030215829 A1	Nuclear hormone receptors
18	US 20030212256 A1	Proteins and nucleic acids encoding same
19	US 20030207854 A1	Antiprogestins with partial agonist activity
20	US 20030186385 A1	Method of identifying polypeptide monobodies which bind to target proteins and use thereof
21	US 20030186313 A1	Methods and compositions in breast cancer diagnosis and therapeutics
22	US 20030143632 A1	Novel genes encoding proteins having prognostic, diagnostic, preventive, therapeutic and other uses

	Document ID	Title
23	US 20030134865 A1	Modulation of histone deacetylase
24	US 20030124598 A1	PGC-1beta, a novel PGC-1 homologue and uses therefor
25	US 20030118997 A1	Human cDNAs and proteins and uses thereof
26	US 20030114517 A1	Substituted urea neuropeptide Y Y5 receptor antagonists
27	US 20030092119 A1	Nuclear hormone receptors
28	US 20030082642 A1	Method and compositions for monitoring DNA binding molecules in living cells
29	US 20030077664 A1	Methods of screening for compounds that modulate hormone receptor activity
30	US 20030027778 A1	Methods and compositions in breast cancer diagnosis and therapeutics
31	US 20020187953 A1	Estrogen receptor-related receptor alpha (ERRalpha) and cartilage formation

	Document ID	Title
32	US 20020165223 A1	Substituted urea neuropeptide Y Y5 receptor antagonists
33	US 20020164578 A1	Nucleic acid molecules encoding glutx and uses thereof
34	US 20020156054 A1	Synthesis and use of retinoid compounds having negative hormone and/or antagonist activities
35	US 20020151588 A1	Dissociated glucocorticoid receptor antagonists for the treatment of glucocorticoid associated side effects
36	US 20020137209 A1	Methods of dissociating nongenotropic from genotropic activity of steroid receptors
37	US 20020119499 A1	Method for identifying or screening agonist and antagonist to PPAR
38	US 20020112251 A1	Novel genes encoding proteins having prognostic, diagnostic, preventive, therapeutic and other uses

	Document ID	Title
39	US 20020106727 A1	Transcriptional intermediary factor-2
40	US 20020086354 A1	Novel genes encoding proteins having prognostic, diagnostic, preventive, therapeutic and other uses
41	US 20020052032 A1	32142, 21481,25964, 21686, novel human dehydrogenase molecules and uses therefor
42	US 20020042371 A1	32142, 21481, 25964, 21686, novel dehydrogenase molecules and uses therefor
43	US 20020037514 A1	Identification of nuclear receptor-dependent coregulator recruitment
44	US 6770449 B2	Methods of assaying receptor activity and constructs useful in such methods
45	US 6662113 B1	Digital correlation of test samples and the screening of interactions between the same
46	US 6627423 B2	21481, a novel dehydrogenase molecule and uses therefor
47	US 6613555 B2	32142, 21481, 25964, 21686, novel human dehydrogenase molecules and uses therefor

	Document ID	Title
48	US 6605627 B2	Modulators of peroxisome proliferator activated receptor-gamma, and methods for the use thereof
49	US 6521624 B1	Synthesis and use of retinoid compounds having negative hormone and/or antagonist activities
50	US 6511834 B1	32142,21481,25964,21686, novel human dehydrogenase molecules and uses therefor
51	US 6469028 B1	Synthesis and use of retinoid compounds having negative hormone and/or anatgonist activities
52	US 6455300 B1	Method and compositions for monitoring DNA binding molecules in living cells
53	US 6365361 B1	Method for identifying or screening agonist and antagonist to PPAR
54	US 6346374 B1	Nucleic acid molecules encoding GLUTX and uses thereof
55	US 6268173 B1	Polynucleotide encoding transcriptional intermediary factor-2

	Document ID	Title
56	US 6228848 B1	Synthesis and use of retinoid compounds having negative hormone and/or antagonist activities
57	US 6218128 B1	Methods of identifying compounds having nuclear receptor negative hormone and/or antagonist activities
58	US 6174682 B1	Thioredoxin family active site molecules and uses therefor
59	US 6147275 A	Corticotropin releasing factor receptor 1-deficient mice
60	US 6136547 A	Nucleic acid molecules encoding glutx and uses thereof

	Document ID	Title
61	US 6090810 A	Synthesis and use of retinoid compounds having negative hormone and/or antagonist activities
62	US 6068976 A	Modulators of ob gene and screening methods therefor
63	US 6008204 A	Synthesis and use of retinoid compounds having negative hormone and/or antagonist activities
64	US 5958954 A	Synthesis and use of retinoid compounds having negative hormone and/or antagonist activities
65	US 5952345 A	Synthesis and use of retinoid compounds having negative hormone and/or antagonist activities

	Document ID	Title
66	US 5942398 A	Nucleic acid molecules encoding glutx and uses thereof
67	US 5919802 A	Methods of preventing and/or treating temporal lobe epilepsy
68	US 5877207 A	Synthesis and use of retinoid compounds having negative hormone and/or antagonist activities
69	US 5776699 A	Method of identifying negative hormone and/or antagonist activities
70	US 5714595 A	Mechanism-based screen for retinoid X receptor agonists and antagonists
71	US 5700682 A	Mechanism based screen for retinoid X receptor agonists and antagonists

	Document ID	Title
72	US 5700650 A	Mechanism-based screen for retinoid X receptor agonists and antagonists
73	US 5691196 A	Recombinant nucleic acid containing a response element
74	US 5648248 A	Methods for producing differentiated cells from immature hematopoietic cells
75	US 5506102 A	Methods of using the A form of the progesterone receptor to screen for antagonists of steroid intracellar receptor-mediated transcription
76	US 5929058 A	Treating symptoms of non-insulin dependent diabetes mellitus - using a combination of glucocorticoid receptor I agonist and glucocorticoid receptor II antagonist

	Document ID	Title
1	US 20030082642 A1	Method and compositions for monitoring DNA binding molecules in living cells
2	US 20020151588 A1	Dissociated glucocorticoid receptor antagonists for the treatment of glucocorticoid associated side effects
3	US 6455300 B1	Method and compositions for monitoring DNA binding molecules in living cells
4	US 5929058 A	Treating symptoms of non-insulin dependent diabetes mellitus - using a combination of glucocorticoid receptor I agonist and glucocorticoid receptor II antagonist